PAIN THEORIES – A REVIEW FOR APPLICATION IN ATHLETIC TRAINING AND THERAPY


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Knowledge is an important tool in treating pain. With the knowledge of pain etiology, the pathways that carry the message of pain to the brain, the social and psychological aspects of pain perception, and the ways in which pharmaceutical agents block pain, athletic trainers and therapists can communicate pain-management options to their athletes. In the daily course of the job, we must deal with our athletes’ pain in order to treat and manage their sports-related injuries. Disregarding an athletes’ pain in the injury-management process can retard rehabilitation.

The objectives of this article are to review three current and well-accepted pain theories, provide ideas to clinicians who are teaching injured athletes about pain, and apply the theories to athletic training and therapy practice in traditional and nontraditional patient treatment.

Pain has been defined as
an unpleasant sensory and emotional experience arising from actual or potential tissue damage or described in terms of such damage...[P]ain includes not only the perception of an uncomfortable stimulus but also the response to that perception (Thomas, 1997, p. 1387).

Pain perception is the body’s way of signaling danger but also can often hinder rehabilitation, activities of daily living, and athletic activities.

Gate-Control Pain Theory

Melzack and Wall’s gate-control theory of pain is the first of the three pain theories reviewed. Within the spinal cord lies a group of nerve cells known as the substantia gelatinosa, also referred to as Laminae II and III of the dorsal horn. Using a gate as an analogy, the substantia gelatinosa is a “gate keeper” that determines whether a sensory message will reach the brain or be blocked. The gate plays an important role in pain management of the central nervous system (CNS). Pain messages that pass through the gate stimulate T cells (transport cells) of the dorsal horn in the spinal cord and facilitate message transport to the brain. T cells send messages up the spinal cord, much as an elevator transports people to the top floor of a building.

The gate-control theory is predicated on transporting messages to the brain via impulses that are received on a “first come, first served” basis, and the strength of the incoming impulses also seems to dictate their influence on the gate. For example, if a 20-lb dumbbell is dropped on the thumb, the weight contacts the thumb and a pain stimulus is created at the pain receptors (nociceptors) in the skin. The pain-carrying A-delta nerve fibers in the peripheral nervous system are activated first and start the message (bright, local pain) moving toward the spinal cord, at a rate of approximately 15 m/s. Activation of C nerve fibers sends a slow pain message (slow, dull, diffuse pain; conducting speed no faster than 2 m/s) that follows the same pathway. The pain messages proceed to the substantia gelatinosa, where substance P is released. The gate is opened and the message is sent to the T cell, which transports the message in the “elevatorlike” spinothalamic or spinoreticular tract in the spinal cord. The message ascends to the reticular formation, which is the part of the brainstem that influences alertness, waking, sleeping, and reflexes (Gilman & Newman, 1992). When the
reticular formation is stimulated, autonomic motor and sensory responses are quickly produced (Buxton, 1999).

This explains why athletic trainers and therapists are taught to pull an athlete’s arm hair, press on the nail bed, or grind their knuckles into the sternum to assess the condition of a potentially brain-injured athlete. If the reticular formation is unharmed, the pain reflex results in a motor action (pulling the arm or finger away or becoming alert) in response to the painful stimulus. The pain threshold determines the first pain felt and is a function of the reticular formation. Once the reticular formation relays the message, the stimulus travels on to synapse with other structures of the midbrain and central brain (Buxton).

The central brain, or cortex, perceives the sensory message of sharp pain (caused by the dumbbell’s effect) on the left thumb and sends an efferent message back down the spinal cord in the “elevator”, which is now descending. Multiple messages to the descending tract can either exaggerate or inhibit the perception of pain. Muscle movement is the result of message facilitation resulting in shaking the left hand or rubbing or massaging the left thumb. Consequently the receptors in the thumb have a new message; rubbing the thumb now sends a message of pressure. The new message is sent along a different pathway, the afferent A-beta nerve – a large, myelinated nerve with a low threshold for stimulation. The A-beta nerve moves the message so fast (up to 70 m/s) that it will reach the gate in the substantia gelatinosa more quickly than the next message of pain from the thumb (Gilman & Newman, 1992). The new message of pressure occupies the gate, and the next message of pain cannot get through the gate to the T cell. As long as the thumb is being rubbed, pressure rather than pain is perceived. When the rubbing stops, the message of pressure no longer occupies the gate and the message of pain is allowed to move to the brain, where pain will be perceived and analyzed. This is, of course, a simplification of a complex series of events. There are many gates in the spinal cord, and all would have to be blocked to cancel pain. At best, rubbing the thumb decreases the pain sensation rather than eliminating it. In short, the gate-control theory of pain states that nonpainful stimuli can block painful stimuli (Buxton, 1999). The perception of pain depends on whether the dominant message is the one ascending to deliver the message of pain or the descending one, which will act to inhibit the painful stimuli (Watt-Watson, 1999).

Acute pain serves an important function because it often protects the body from further harm. When pain continues past the protection and warning phase, it becomes problematic. Chronic pain, pain that lasts longer than 6 months postinjury, can also be explained by the gate-control theory (Prentice, 1999). Through a negative-feedback system in the spinal cord, the gate remains open, with less input from the large A-beta afferent nerves and more input from the small pain-carrying sensory nerves (Buxton, 1999). Overriding of small pain-carrying nerves by large afferent nerves is the foundational theory behind the use of transcutaneous electrical nerve stimulators (TENS) as a pain-modulating tool (Figure 1). TENS stimulates the afferent nerve fibers that travel along large myelinated nerves to the substantia gelatinosa that occupy the gate, thus blocking the painful stimuli traveling along the smaller unmyelinated nerve fibers to the gate. Ice application and electrical stimulation work by stimulating sensory nerves in the skin to produce pain relief. The applications of the gate theory account for the effectiveness of analgesic balms and other counterirritants in changing the perception of pain (Prentice). Some joint-mobilization techniques are believed to stimulate mechanoreceptors and block nociceptive pathways (Kisner & Colby, 1996). In practical experience, the gate theory seems to be valid.

**Central-Biasing Pain Theory**

Chronic pain can also be a learned behaviour of patterned pain (Buxton, 1999). The central-biasing, or central-control-mechanism, theory can explain the concept of a “learned behaviour” (Starkey, 1993). This theory builds on the gate theory (acting within the spinal cord) and addresses brain influences on
incoming and outgoing messages. Cognitive effects can alter sensory discrimination, the location of the pain source, the intensity of the pain, or the nature of the pain (Prentice, 1999). Referred pain can be explained as a location error by the brain. As sensory nerves branch with, or cross, other afferent nerves in the spinal cord, pain can be displaced or misplaced and thus referred to another area of the body (Buxton). Referred myofascial pain is caused by the depolarization of neurons in many spinal segments that are distal to the pain origin. Referred pain can help athletic trainers and therapists find the source of painful stimuli. For example, pain that radiates down the left arm is known as Kehr’s sign and suggests spleen pathology or heart attack.

The central-biasing mechanism explained by Meizack and Wall also has motivational-affective influences. An internal drive or external stimulation can have a strong influence on thought processes and, therefore, the affect or perception of pain. The ascending message of pain passes through the reticular formation, the thalamus, and might enter the limbic system. This higher brain system functions to link motivational and emotional responses to the pain message (Gilman & Newman, 1992). The motivational affective mechanism of the limbic system explains emotional responses to pain, fear, rage, crying, panic, denial, and anxiety. A visceral response to pain might accompany the emotional reaction, causing nausea immediately after a painful experience. A fearful or angry response to pain also explains the reaction of an athlete who screams. “Don’t touch me!” Once such athletes have been calmed and comforted, they shake it off and report that they were not in pain but were scared -- quite a normal and valid response to injury.

On a subconscious level, mental state or personality can affect the response to a pain message (Starkey, 1993). Some athletes in pain continue to play because the motivation of fear or punishment is greater than the motivation to protect the injured tissue. Some athletes might be afraid of quitting and resist an injury. At the beginning of a season an athlete might tolerate more pain than at the end of the season, when he or she is fatigued. Perhaps this is why some athletes are considered to be “tough” while others give up easily.

“Pain tolerance is the maximum degree of pain intensity a person is willing to experience” (Watt-Watson, 1999, p. 325). Athletic trainers and therapists and coaches often speak of athletes’ pain tolerance — what makes athletes tough, what allows them to play in pain, or perhaps what makes an athlete a “wimp.” Tolerance discussions should include consideration of the cortex of the athlete’s brain, the limbic system, reticular formation, and the thalamus. These brain structures interpret the intensity of the pain, send autonomic responses, regulate pain thresholds, and modify the emotional response to pain. Pain response appears to be more physiological than psychological in origin (Watt-Watson).

The pain-detection threshold is both biological and subjective and thus has many influential denominations. Influences on pain tolerance include drugs, alcohol, and hypnosis, any of which can alter the cognitive function of the brain and increase tolerance. Faith and strong beliefs can enhance pain tolerance. Warmth, rubbing, and distraction have also been thought to increase tolerance of pain. Pain tolerance can be reduced by fatigue, anger, boredom, anxiety, stress and depression. Persistent pain also seems to decrease tolerance (Watt-Watson, 1999). Because of the varied and multiple factors that can decrease pain tolerance, athletes who do not respond well to therapy should not be judged as malingerers or “fakers”. Disregarding the physiological effects of psychological influences creates an incomplete picture of pain etiology and is an injustice to the athlete.

The central-biasing theory also explains cognitive evaluation such as conscious thought (Starkey, 1993) and past experiences or pain memory (Prentice, 1999). An athlete who cringes and grimaces before a painful bruise is actually touched exemplifies pain memory. We often observe athletes who, after return to play, overreact to a situation similar to that involving a previous injury. Past experiences and learned behaviors might explain the sociological factors associated with central biasing. Picture the Little League shortstop who is hit with a batted ball in the groin. If the athlete is a boy, he might react to pain much differently than a female gymnast who falls on the uneven bars and injures her groin. This is an example of gender-role stereotypical response. Much research has been conducted comparing the pain response between genders (Buxton, 1999).

Another example of the influence of conscious thought on pain tolerance is peer pressure (Buxton, 1999). An athlete who changes a pain complaint after a pep talk from a team mate is consciously overriding pain (Starkey, 1993). Culture, ethnicity, socioeconomic status, the type of sport played (contact vs. noncontact), personality type, and being in the “heat of the game” all influence pain tolerance (Buxton; Prentice, 1999).

Central-biasing theory might be related to placebo effects of pain relief. The etiology of a decrease in pain without physiological change to the issue could be interpreted as a psychological mechanism. “All therapeutic modalities have some degree of placebo effect” (Buxton, 1999, p.59). For example, an athlete might discuss a great new modality that has helped him or her achieve pain-free
range of motion when, in fact, the ultrasound machine used on that athlete had a broken crystal. The central-biasing theory is applied to athletic training or therapy when we ask an athlete to describe and rate his or her pain on a scale of 1 – 10 (Buxton). Athletic trainers and therapists working with athletes in pain should capitalize on the central-biasing theory by initially calming and comforting the athlete, empathizing once the athlete is calm, and justifying the reaction that he or she is having to the painful experience. Education and behaviour modification can give the athlete confidence and assurance that pain is a protective process and that there are many ways of managing it.

Endogenous-Opiates Pain Theory

Castel introduced a theory of pain after endogenous opioids were discovered in the human body in the 1970s (Prentice, 1999). The body produces three types of peptides that have opioid-like properties: enkephalins, endorphins, and dynorphins (Lehne, 2001). To review this pain theory, the initial tissue response to injury and descending neural impulses must be examined. When tissue is subjected to injury, bradykinin, prostaglandin, and substance P are released into the tissue. These mediators influence the initial inflammatory response to injury. Bradykinin directly produces painful stimuli. Prostaglandin and substance P, a type of neurotransmitter, lower the nerve’s threshold to bradykinin, which magnifies the perception of pain. Repeated or prolonged stimuli perpetuate the continued feeling of pain (Buxton, 1999). In the spinal cord, opioid peptides are released to modulate pain transmission (Gilman & Newman, 1992). The enzyme cyclo-oxygenase catalyzes the formation of prostaglandin. The blocking of prostaglandin synthesis by nonsteroidal anti-inflammatory drugs (NSAIDs) is the base for using these drugs to control pain and inflammation. Over time, these chemical irritants released into the tissue diffuse, making a larger area hypersensitive to pain perception (Buxton), explaining why pain becomes less localized over time in an acute stage of inflammation, making pinpointing pain later in the healing process difficult.

Methionine enkephalin also influences the release of pain modulators. Enkephalins are natural endogenous opiates found in the CNS that are released rapidly for short-term pain relief (Buxton, 1999; Prentice, 1999). They counteract the effect of substance P in the substantia gelatinosa and thus inhibit the transmission of painful stimuli to the T cell (Watt-Watson, 1999). Endorphins originate from the hypothalamus, which is where pain threshold is influenced (Buxton).

The thalamus directs the sympathetic nervous system to gear up for “fight or flight” which involves the release of norepinephrine/adrenaline. Beta endorphins are released in the cerebral spinal fluid in the presence of severe stress and physical exertion, a process that is believed to be associated with the so-called runner’s high. Just as morphine is an addictive drug, endorphins can create a strong desire in the runner to continue to run with pain until the “high” is achieved. Endorphines can also be released through high voltage and low frequency electrical stimulation protocols that inhibit noxious stimuli. Beta endorphins can provide up to 4 hours of natural pain relief (Starkey 1993)

Integration and Application of Pain Theories

Integrating the three pain theories can explain an athlete’s behaviour and response to pain. Using trigger points as an example, pain theories can be integrated and applied to pain management strategies. Pain receptors at trigger points are hypersensitive to pain, and the pain message is initiated when depolarization of the receptors occurs. The message moves through the spinal cord, the gate, and to the brain. The brain detects a dull, aching chronic pain. One way to manage chronic pain is to relate the gate control theory of pain to modality choice. TENS (Figure 2), massage, and analgesic balms stimulate non-pain-afferent fibres; ice decreases the pain-afferent-fibre velocity – all are used to block pain sensation at the gate. Another pain management technique is to use the central blasting theory to decrease anxiety, stress, and fear by educating the athlete on the natural process of healing and pain relief. A third strategy is to use CNS opiates by pressing on the trigger point so hard (acupressure) that the pain is increased to nearly intolerable levels, which in turn stimulates the release of beta endorphins.
Other modality strategies to manage chronic pain caused by muscle or connective tissue injury include the use of “electroacupuncture and possibly TENS, with long phase durations and low pulse rates (1 to 5 pulses per second) to cause small diameter fiber depolarization necessary for (beta endorphin) release” (Prentice, 1999, p.32). Another approach is the use of drugs to relieve pain by blocking prostaglandin synthesis in the damaged tissue or to slow or block the impulses travelling on A-delta or C fibers (Prentice). NSAIDS are used to block prostaglandin synthesis to mediate pain at the injury site. Morphine and morphineline drugs - Demerol®, Darvon®, and codeine - mimic endogenous opiates by binding to receptors in the CNS (Lehne, 2001)

Pain management begins with identifying the source of pain, decreasing the chemical and mechanical causes, and facilitating tissue healing (Prentice, 1999). A single stimulus or a combination of many stimuli can trigger the pain response, but a person’s reaction to pain cannot be measured objectively. Pain is a learned and complex experience and therefore requires multiple strategies for management (Watt-Watson, 1999). Physical, behavioural, and cognitive methods can all be used by athletic trainers and therapists to help athletes manage pain (Buxton, 1999). Therapeutic modalities, medications, exercise, and surgery are examples of tools used to facilitate tissue healing and reduce pain. Manual therapies such as muscle energy, massage, and myofascial release are non-traditional ways to manage pain. Producing a reward system, using distractions such as talking with an athlete during treatment, using relaxation techniques, and decreasing fear and anxiety are examples of behavioural tools to manage pain (Starkey, 1993; Prentice). Empathy and understanding are prerequisites to help athletes in pain (Watts-Watson). Cognitive methods include thorough education and explanation of pain and injury processes.

The American Pain Society has suggested that health-care workers assess pain as the fifth vital sign, along with heart rate, blood pressure, respiration, and temperature (McCaffery, 2001). Knowledge is the key to successful management of pain associated with athletic injuries. By reviewing the neuroanatomy, understanding the pain theories, and applying the theories to pain management, we can facilitate pain relief for our athletes.

References


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